

Effect of Previous Hamstring Strain on Anterior Cruciate Ligament (ACL) Forces

By

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Introduction: Anterior Cruciate Ligament (ACL) injuries are very common in the athletic population. While the hamstring muscles help protect the ACL, hamstring strain injuries (HSI) are also common in sports. After HSI, the muscle goes through many different adaptations that often alter the overall function of the hamstrings. Therefore, it is possible that the associated deficits to hamstring muscle function after HSI may alter ACL loading.

Methods: Nine subjects were included in the study. Six in the healthy control group, and three in the previous HSI group. We used an ultrasound based subject-specific musculoskeletal model to estimate muscle forces and ACL forces during walking, squatting, and landing from a jump. Ultrasound data, along with static calibration from motion capture, were used to develop the subject specific model. The purpose of this study was to determine if people with a previous hamstring strain had increased ACL forces during common activities of daily living and sport compared to healthy controls. We hypothesize that people who have had a previous hamstring strain injury will have increased forces on the anterior cruciate ligament compared to individuals with no history of hamstring injury.

Results: Our results did not support our hypothesis. People with a previous HSI did not have increased ACL forces compared to healthy controls. **Discussion:** Three subjects in the previous HSI group move differently compared to the control group. In three of the four dynamic tasks, the previous HSI subjects demonstrated significantly less sagittal plane hip range of motion compared to healthy controls. We also found significant differences in hamstring EMG during the drop jump task. We concluded that people may move differently after sustaining a HSI to compensate and ultimately decrease their risk of further injury.

Effect of Previous Hamstring Strain on Anterior Cruciate Ligament (ACL) Forces

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Chapter I: Introduction

Anterior Cruciate Ligament (ACL) injuries are common in the active population around the world.¹ ACL injuries often occur during unplanned side cutting maneuvers when there is increased anterior tibial translation and valgus collapse.² Short-term effects of ACL injury often include surgery and a long recovery period before returning to sports. Long-term effects have been linked to knee osteoarthritis (OA).³ The risk of developing knee OA after ACL injury has been reported to be as high as 50%.⁴ Complete tears of the ACL result in a higher chance of developing knee OA compared to a partial tear.⁵ Nevertheless, developing knee OA after ACL injury may occur sooner than normal when compared to the normal aging population. Therefore, protecting the ACL from injury has critical significance in the active population.¹

The ACL provides a posterior pull onto the tibia when an anterior load is applied. The hamstring muscles, when properly functioning, are protective of the ACL.⁶⁻⁸ The shear component of the hamstring force provides a posterior pull on the tibia, thus providing supplementary ACL protection.⁹ Cadaver studies that measure ACL loading directly have shown statistically and clinically significant data to support that the hamstrings are protective of the ACL.^{7,8} For example, Markolf et al (2004) found that when applying one hundred newtons of force to the hamstring of a cadaver knee during passive extension, ACL force decreased significantly compared to no muscle loads. The decrease in ACL loading happened mainly between 0-60 degrees of flexion, with differences in ACL forces between 5-60 N.⁷ Since the hamstrings are protective of the ACL, hamstring function is extremely important. If hamstring muscle function is altered due to injury, its capacity to protect the ACL may be compromised.

Hamstring strain injuries (HSI) have been linked to several adaptations that may alter hamstring function. Hamstring strains are common sports injuries that often occur when

sprinting.¹⁰⁻¹² Although HSI can occur in different locations of the hamstring musculature, they most commonly involve the proximal musculotendon junction of the biceps femoris long head.

13-15

Several neuromuscular adaptations have been linked to athletes with a history of HSI such as; lower eccentric knee flexor strength^{16,17}, lower voluntary myoelectrical activity during maximal knee flexor eccentric contraction^{18,19}, lower knee flexor eccentric rate of torque development²⁰, lower voluntary myoelectrical activity during the initial portion of eccentric contraction^{16,20}, and lower functional hamstrings-to-quadriceps ratio.^{6,16} Persistent strength loss may also be an effect of atrophy in the previously injured muscle.¹³ The presence of scar tissue is common in many people with previously strained hamstrings.²¹ It is theoretically plausible that the presence of scar tissue may alter in-vivo muscle contraction mechanics, although, this is not well supported in the literature. Many adaptations have been reported to decrease hamstring function after HSI. Based on prior research that people experience functional deficits after sustaining hamstring strain injuries, it is possible that ACL forces are increase because of this.

Hypothesis

We hypothesize that people who have had a previous hamstring strain injury will have increased forces on the anterior cruciate ligament compared to individuals with no history of hamstring injury during common activities of daily living and sport.

Purpose

The purpose of this study is to compare ACL forces during common activities of daily living and sport in people with a previous hamstring strain compared to healthy controls.

Delimitations

- 1) The subjects were between 18-25 years old.
- 2) The subjects were recreationally active, as defined by ACSM (150 minutes per week of aerobic exercise, and 2-3 days per week of resistance training)
- 3) The subjects in the hamstring injury group had past self-reported or diagnosed hamstring strain that did not require surgery. Grade 1 or 2.
- 4) The subjects in the hamstring injury group had returned to an active state or cleared by a health care professional to return to sport
- 5) The study is limited to analyzing 4 movements; squatting (double and single leg), drop jump, and walking. These movements mimic common motions of daily living and sport.

Limitations

- 1) ACL forces will be estimated based on inverse dynamics and muscle force calculations.
- 2) The EMG signals recorded during data collection are manipulated during the optimization process

Chapter II: Literature Review

Introduction

Lower extremity injuries are common among the active population worldwide.¹ Specifically, anterior cruciate ligament (ACL) injuries are common in many sports and other activities that involve high impact forces.¹ ACL tear is a serious knee injury that involves surgery and a long recovery period. Griffin et al. reported an incidence of 80,000 ACL injuries per annum in the U.S.A.²² Anterior cruciate ligament injury in athletic populations are more common than in the general population²³⁻²⁵, and they occur more commonly in field and court sports.^{26,27} ACL reconstructive surgery is common among people who have suffered a complete tear. Allografts or autographs are used to reconstruct the ACL surgically. This surgery involves a long-term recovery and rehabilitation period. Long-term effects of ACL injury include the development of knee osteoarthritis (OA). Barenius et al. (2014) found that people with prior ACL injury are three times more likely to develop knee OA later in life compared to contralateral healthy knee. Based on the short and long-term consequences of sustaining an ACL tear, efforts to protect the ACL from initial injury are critical.

The hamstrings are protective of the ACL. Hamstrings provide supplementary protection of the ACL, specifically from the shear component of hamstring force providing posterior pull of the tibia. Since ACL injury has been linked to increased anterior tibial translation, the hamstrings provide an extremely important role in counteracting this force. Hamstring strain injuries are shown to lead to many functional deficits that may, in turn, alter the loading on the ACL.

During this review, the function of the hamstring muscle will be investigated, and

specifically, how it affects the ACL. Then, an elaboration of the adaptations that occur after a hamstring strain injury will be investigated. Lastly, evidence that suggests previous hamstring strain leads to increased risk of ACL injury will be presented.

The Hamstrings effect on the Anterior Cruciate Ligament (ACL)

Mechanism of ACL Injury

The Anterior Cruciate Ligament (ACL) is a stabilizing knee ligament. Its function is to resist excessive anterior movement of the tibia in relation to the femur. Also, it limits rotational movement of the knee. ACL injury typically occurs at foot plant with a low knee flexion angle, knee joint rotation, and valgus collapse.² This kinematic profile results in high anterior shear forces and, increased anterior tibial translation, which increases ACL strain.²

Cadaver Studies

The hamstrings are protective of the ACL. The shear component of hamstring force provides posterior pull on the tibia, thus providing supplementary ACL protection.²⁸⁻³⁰ Through compressive and posteriorly directed shear forces, the hamstrings provide dynamic knee stability by limiting anterior tibial translation³¹ and torsional loading.⁹ In an in-vitro cadaver study by Withrow et al. (2008), they examined the effects of the hamstring and quadriceps muscles on relative ACL strain. A differential variable reluctance transducer was placed on the ACL of eighteen cadaver limbs to measure relative strain. Three different loading patterns were conducted on each cadaver limb to simulate a jump-landing pattern. Constant hamstring force (isotonic), increasing hamstring force (lengthening), and absence of hamstring force were performed and ACL relative strain was measured during each trial. The results from this study found that increasing hamstring muscle force reduced peak relative ACL strain by 70%,

specifically during the lengthening contraction.⁸

Another factor shown to affect ACL loading is the relative quadriceps to hamstring force ratio. Increased anterior tibial translation often occurs when there is reduced activity of the hamstrings relative to the quadriceps.⁶ This activity reduces the knee flexion angle and therefore, increases ground reaction force that passes through the knee joint. This increases shear force of the femur over the tibia, therefore there is an increase in anterior tibial translation. In a study done by Markolf et al. (2004), thirteen cadaveric knee specimens had load cells installed to record resultant forces in both anterior and posterior cruciate ligaments under 5 loading conditions. The tibial loading conditions were, no tibial force, 100-N posterior tibial force, 100-N anterior tibial force, 5-Nm internal tibial torque, and 5-Nm external tibial torque. The results showed that the hamstrings reduce ACL loads and the quadriceps increase ACL loads depending on the knee flexion angle. At 20 degrees of flexion, ACL force decreased by 20% during hamstring loading compared to no muscle loads. At 40 degrees ACL force decreased by 77% and at 60 degrees it decreased by 120%. It was also found that the hamstrings essentially negate ACL force produced by an applied 100-N anterior tibial force beyond approximately 60° of flexion.⁷ Although ACL injuries often occur at small flexion angles, this small reduction in ACL force produced by the hamstrings could potentially be the difference between a tear or, no tear.

Hamstring Activation Patterns and its Effect on ACL Loading

The balance of muscle activation between the hamstring and quadriceps play an essential role in the prevention of increased ACL strain.³²⁻³⁴ Cowling and Steele (2000) suggested that hamstring activation patterns before initial contact in land and stop movements are preplanned to counter ACL loading at landing.³⁵ Likewise, Besier et al. (2003) found that activation patterns

are preplanned to counter loading in varus/valgus and internal/external moments at the knee.³² Therefore, the neuromuscular function of the hamstring muscle group is critical to reduce ACL loads.² In a cohort study by Zebis et al. (2009), fifty-five female handball and soccer players with no prior history of ACL injury were tested for EMG pre-activity. Pre-activity meaning the EMG readings prior to initial contact of side-cutting maneuver. The vastus medialis and lateralis, rectus femoris, semitendonsis, and biceps femors were tested during a standardized side-cutting maneuver. Sports related injuries were documented on all players for two seasons. A total of five ACL tears occurred during that time. The results showed those who sustained ACL injury had reduced EMG preactivity of the semitendonsis and increased EMG preactivity of the vastus lateralis during side cutting.³⁴ Increased preactivity of the quadriceps muscles compared to the hamstrings may increase the risk of ACL injury. However, these studies were done specifically on women because of the high incidence rate of ACL injury compared to men. The effects of EMG preactivity on ACL injury has yet be determined in men.

After giving explanation and evidence that the hamstrings reduce ACL loads, it can be implied that proper hamstring function may be critical in protecting against ACL injury since all previous studies utilized healthy limbs. If hamstring function is altered, it can alter overall forces acting on the knee joint. Altered hamstring function has been linked to negative adaptations after sustaining a hamstring strain injury.⁶

Hamstring adaptations after Hamstring Strain Injury

Hamstring strain injuries (HSI) are common in sports that involve sprinting.¹⁰⁻¹² Although HSI can occur in different locations of the hamstring muscle complex, they most commonly involve the proximal musculotendon junction (MTJ) of the biceps femoris long head (BFLH).^{13-15,36} HSI are thought to occur during the late swing phase of sprinting^{37,38} and during the active

lengthening contraction.^{39,40} Many studies have shown re-injury of 15%^{11,41-43} to more than 50%^{44,45} when athletes return to sport before their rehabilitation is complete.⁴⁶ Proper rehabilitation is critical in all sports injuries. However, long-term studies showing the effects of specific rehabilitation techniques after hamstring strain injury are limited. That being said, eccentric strengthening is believed to increase the series compliance of muscle and allow for longer operating lengths and potentially reduces the risk of injury.^{47,48}

Croisier et al. (2002) found a significant difference in the quadriceps to hamstring strength ratio in athletes with previous HSI compared to uninjured athletes. Also, 18 of the 26 subjects had strength deficits in the previously injured hamstring. Specifically, an 11% reduction in concentric peak torque and a 22% decrease in eccentric torque.¹⁶ When comparing 2 rehabilitation programs, Sherry et al. (2004) found that a rehabilitation program including progressive agility and trunk stabilization exercises are more effective than a program emphasizing isolated hamstring stretching and strengthening in promoting return to sports.⁴⁹ These findings suggest that neuromuscular control of the hamstrings is altered after injury. Although rehabilitation techniques may be an important factor that contributes to returning muscle to normal function, it appears that the hamstrings have an incomplete healing response after HSI based on previous research.

Neuromuscular changes associated with HSI

Compared to healthy individuals, several neuromuscular adaptations have been linked to athletes with a history of HSI such as; lower eccentric knee flexor strength^{16,17}, lower voluntary myoelectrical activity during maximal knee flexor eccentric contraction^{18,19}, lower knee flexor eccentric rate of torque development²⁰, lower voluntary myoelectrical activity during the initial portion of eccentric contraction^{16,20}, and lower functional hamstrings-to-quadriceps ratio.^{6,16}

Persistent strength loss often occurs after HSI. For example, Lee et al. (2009) found significant weaknesses in eccentric hamstring strength in the limb with prior HSI compared to the uninjured limb (14%).¹⁷

Sole et al. (2011) collected EMG and dynamometer strength data between previous hamstring strain injury participants and healthy controls. They found a significant decrease in eccentric flexor torque of the injured group between 5 and 25 degrees of knee flexion when compared to the uninjured group. Also, they found significantly less EMG activation of the hamstrings in the previously injured subjects compared to control group. These significant differences were seen between 5-65 degrees during eccentric contraction trials. These findings suggest that muscle activations may have a predominant impact contributing to decreased torque and agonist hamstring muscle activity in the lengthened range of eccentric flexor contractions in participants with a recent history of a hamstring injury.⁵⁰ Similarly, Opar et al. (2013) found that previously injured hamstrings displayed lower rate of torque development and impulse during slow maximal eccentric contraction compared with the contralateral uninjured limb (39%-40%).⁵¹

Structural Adaptations after HSI

A natural consequence of previous HSI is persistent strength loss¹⁶. Strength loss may also be an effect of atrophy in the previously injured muscle.¹⁶ In a longitudinal study, 50% of athletes were shown returning to sport after HSI having substantial reduction in BFLH volume.¹³ Also, along with this reduction in BFLH volume, hypertrophy of BFSH has been shown in the previous HSI group.¹³ This shows adaptations that may occur after HSI, and is important to note that a change in muscle volume could affect overall muscle function, specifically in strength.

Another factor, which may also alter hamstring muscle function, is the presence of scar tissue. As early as 6 weeks post-injury⁵² and as late as 23 months post-injury, scar tissue has been visible adjacent to the site of original injury.¹³ This is important because it has been shown that scar tissue may alter in-vivo muscle contraction mechanics.¹⁰ Specifically, the replacement of contractile tissue with connective scar tissue could alter force transmission paths through the hamstring⁵³ and change the series stiffness experienced by adjacent muscle fibers. Although there is limited evidence that scar tissue alters force transmission paths throughout muscle, it is theoretically plausible that the presence of scar tissue compromises overall muscle function.

In summary, although lacking substantial longitudinal evidence, it appears the hamstrings go through many changes following hamstring strain injury. Structural, neuromuscular and physiological changes have all been linked to different alterations of the hamstring muscle complex following HSI. It is theoretically feasible that these adaptations lead to altered loading on the joints surrounding the hamstrings. Which, in turn can affect the stabilizing ligaments of the knee, and specifically the ACL.

Supporting Evidence that Hamstring Muscle Dysfunction leads to Increased Risk of ACL Injury

Currently there is not much research that supports the fact that prior hamstring strain injury alters loading on the ACL. However, in a population that is at highest risk for sustaining ACL tears, female athletes, there is evidence also that hamstring muscle function is inadequate thus supporting the “link” between hamstring muscle dysfunction and increased ACL loads.

Blackburn et al. found in females a lower knee flexor rate of force development during

concentric and eccentric isometric contraction compared to men.⁵⁴ Meaning that females take a longer period of time to reach 50% maximal hamstring muscle force when compared to men. These differences in neuromechanical function of the hamstrings may contribute to the greater incidence of ACL injury in females. Similarly, Malinzak et al. (2001) found lower electromyographic hamstring activity during a sidestep cutting maneuver in women. Specifically, women displayed lower EMG amplitude compared men when doing a sidestep cutting maneuver. This may be an important factor because prior research has shown women having a higher incidence rate of ACL injuries compared to men.⁵⁵ Although we will not be investigating sex differences in our study, because females (the population at highest risk for ACL injury) have altered hamstring function, it provides proof of concept that altered hamstring function is related to ACL injury risk.

Musculoskeletal modeling is a common tool used in biomechanics to calculate the forces acting on and inside the body.⁵⁶ Measuring forces and strains on soft tissues have been investigated mainly in cadavers and animals. However, modeling helps to make accurate assumptions of forces and strains through non-invasive data collection techniques.

Loading on the ACL has been investigated in vitro ⁵⁷⁻⁵⁹, in vivo ^{60,61}, and using musculoskeletal modeling techniques ⁶²⁻⁶⁴. The hamstrings are protective of the ACL, and previous studies modeling techniques have supported this. For example, Weinhandl et al. (2014) found that decreased hamstring strength increases ACL loading during anticipated sidestep cutting.⁶⁵ Therefore, we feel confident in using a musculoskeletal modeling technique to predict ACL loading in dynamic tasks of daily living and sport.

Summary

It has been shown that the hamstrings are protective of the anterior cruciate ligament. By providing a posterior pull on the tibia, the hamstrings limit anterior tibial translation, one of the primary knee movements associated with ACL tears. Hamstring strain injuries have shown to decrease the functional capacities of the hamstring muscle complex, such as decreased strength and neuromuscular adaptations.

We hypothesize that people who have had a previous hamstring strain injury will have increased forces on the anterior cruciate ligament compared to individuals with no history of hamstring injury. The purpose of this study is to determine if people with a previous hamstring strain have increased ACL forces during common activities of daily living and sport compared to healthy controls.

Chapter 3: Methods

Design

The purpose of this study was to determine if physically active individuals with a previous hamstring strain had increased ACL forces during common activities of daily living and sport compared to healthy controls.

We hypothesized that people who had a previous hamstring strain injury would have increased forces on the anterior cruciate ligament compared to individuals with no history of hamstring injury. The study followed a cross-sectional design. We utilized a subject-specific musculoskeletal modeling approach in the estimation of muscle and anterior cruciate ligament forces.

Subjects

The subjects used in this study were 9 young adults between 19 and 25 years old (mean age: 21.4 ± 2.0). 5 subjects were male (mean height: $1.80\text{m} \pm 0.03$, mean body mass: $77.0\text{kg} \pm 9.64$) and 4 subjects were female (mean height: $1.61\text{m} \pm 0.04$, mean body mass: $56.8\text{kg} \pm 1.50$). 3 subjects were included in the previous hamstring strain injury group (Table 1), and 6 subjects were in the healthy control group. Subjects in both the healthy and previously injured groups were recreationally active, as defined by ACSM. Subjects in the hamstring injury group had at least one grade 1 or 2 hamstring strain diagnosed by a physician. We did not use specific muscle injury site as inclusion or exclusion criteria. All HSI subjects were self-reportedly cleared to return to normal recreational activity by a physician or certified athletic trainer. In addition, HSI subjects had no other lower extremity injuries, such as ACL or meniscus tears, or acute aches or pains at the time of testing. The control group had no self-reported previous lower extremity

surgery, or muscle strains. Informed consent documents, approved by the UMCIRB, were read and signed by all subjects.

Table 1: Previous Hamstring Strain Injury Subject Table

| HSI Subject | Sex | Height (m) | Mass (kg) | Aprox. Injury Date | Limited | HFQ |
|-------------|-----|------------|-----------|--------------------|-----------|------|
| 1 | M | 1.8 | 87.9 | Feb. 2015 | 3-4 weeks | 86.2 |
| 2 | F | 1.6 | 58.9 | July. 2014 | 1 week | 83.0 |
| 3 | M | 1.8 | 73.0 | Nov. 2012 | 2 weeks * | 92.2 |

Table 1: Previous hamstring strain injury group. HFQ = hamstring function questionnaire score. Scale: 1-100, where 100 indicates subjects reporting no limitations, complaints or symptoms of their hamstring function at time of data collection. * Subject 3 reported that he was limited for two weeks after injury, then stated that his rehabilitation should have been longer because the injury limited his track performance in the spring. Also stated he still had felt lingering effects from the injury on the day of data collection.

Procedure

This study was conducted at the Biomechanics Laboratory inside Ward Sports Medicine building at East Carolina University. Subjects reported to the laboratory for data collection on two separate days. The first day consisted of informed consent (Appendix B), hamstring function questionnaire (for HSI subjects only) and ultrasound imaging. The second day consisted of dynamometer, EMG, and motion capture protocols.

Day 1

Each subject's height and weight were recorded using a Seca 703 digital scale (Seca GmbH & Co. KG, Hamburg, Germany). Ultrasound images were recorded of the biceps femoris long head (BFLH), biceps femoris short head (BFSH), semitendinosus (ST), semimembranosus (SM), vastus lateralis (VL), vastus medialis (VM), vastus intermedius (VI), rectus femoris (RF), medial gastrocnemius (MG), and lateral gastrocnemius (LG)) muscles of either the right or left leg depending on injury site. The right leg was imaged for all control subjects. The ultrasound unit (SuperSonic Imagine, Aixplorer, Bothell, WA) was used along with Aquasonic Ultrasound Gel (Parker Laboratories, Aquasonic 100, Fairfield, NJ) to ensure appropriate coupling to the

skin during scans. All subjects laid prone on a treatment table while images were taken of the hamstrings and gastrocnemius muscles. Subjects laid supine during images of the quadriceps muscles. Images from the ultrasound were used to measure cross sectional areas, fascicle lengths, and pennation angles. Serial cross-sectional images were visualized along the length of the muscle (Figure 1A). Longitudinal (parallel to the muscle belly) images were recorded to determine fascicle lengths and pennation angles (Figure 1C)

Cross sectional area images of the biceps femors long head were recorded from the distal musculotendon junction to the proximal musculotendon junction. (Figure 1B) Once the proximal and distal visible CSA boundaries were identified and marked, ten equidistant marks were made on the skin. Cross sectional images of the BFLH and VL were made at each one of the ten marks running along the muscle belly. Two trials at each location were done for consistency.

Longitudinal panoramic images were recorded along the muscle belly of all muscles (Figure 1D).

Figure 1. Cross Sectional Area and Longitudinal Ultrasound Images

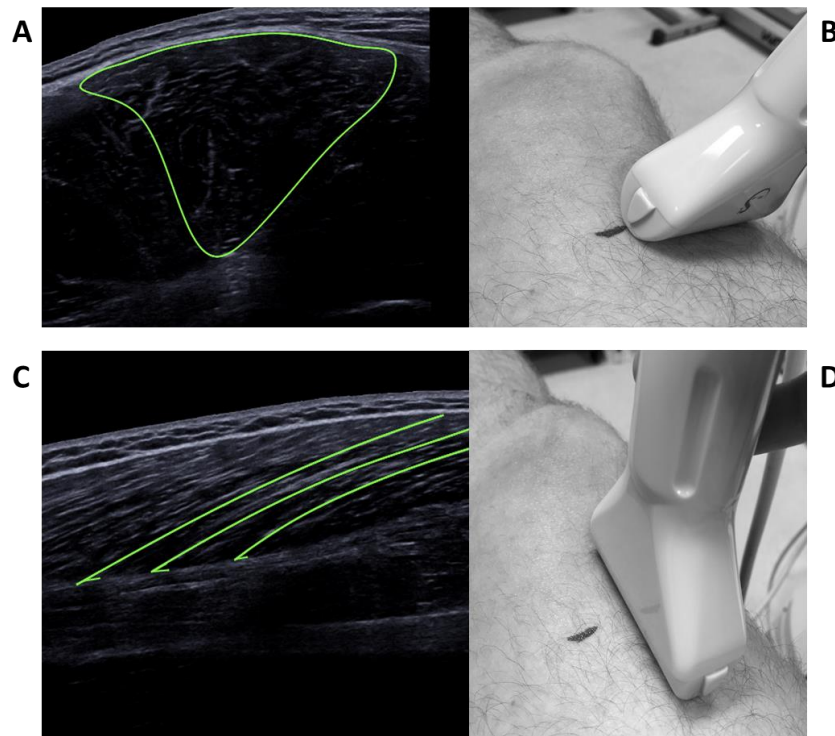


Figure 1: Ultrasound probe orientation (B and D) and images viewed on OsirX (A and C). Note: The ultrasound images are not associated with the adjacent probe image. These images were used to show probe orientation for cross sectional and longitudinal images.

Day 2

A 16 channel wireless EMG system (Delsys Trigno™ Wireless Systems, Delsys®, Natick, MA) collected electromyographic (EMG) data of the medial hamstrings, lateral hamstrings, medial gastrocnemius, lateral gastrocnemius, vastus lateralis, rectus femoris and vastus medialis during the dynamometer protocol and motion capture protocol. The area in which the electrodes were placed were prepped by shaving the area with a disposable razor, and then cleaned using lemon prep abrasive lotion to abrade the skin to lower skin impedance. The skin was then cleaned using alcohol wipes. Qualysis software (Qualisys Medical AB, Gothenburg, Sweden) was used during each dynamometer trial and dynamic motion trial to record the EMG data.

A HUMAC NORM Dynamometer (CSMI, model 502140, Stoughton, MA) was used to test passive and maximal knee flexion and extension strength in both groups. Hip flexion of the subject was set at 90° and the seat was adjusted for each participant. Specifically, the lateral epicondyle of the knee was lined up with the axis of rotation of the dynamometer. 3 consecutive passive torque measurements were taken when the dynamometer moved the knee through the full range of motion at $5^{\circ}/\text{sec}$. 3 repetitions of maximal isometric knee flexion torque were measured at 30° , 45° , and 60° and 3 repetitions of maximal isometric knee extension torque were measured at 60° , 80° , and 100° . The subject was instructed to pull as hard as they could for 5 seconds. 3 repetitions of maximal concentric isokinetic contractions of knee flexion at $60^{\circ}/\text{sec}$ were also completed. The testers used motivation and verbal encouragement to obtain peak torque values from the participant. The subjects were also instructed to try to keep their knee as close to the seat as possible to minimize compensatory movements. Similar verbal encouragement was used during these movements to obtain peak isokinetic torque.

Eight Qualisys ProReflex MCU 240 cameras were used to capture motion on top of a large AMTI force plate (AMTI Model BP6001200-2K, Watertown, MA) and small AMTI force plate (AMTI Model OR6-6-2000, Watertown, MA). For the static calibration trial, reflective markers were placed on the top of the head, lateral epicondyles of the humeri, styloid processes of the radii, anterior superior iliac spines (ASIS), posterior superior iliac spines (PSIS), top of the iliac crests, greater trochanters of the femur, medial and lateral knee at the tibiofemoral joint, at the ankle on the lateral and medial malleoli, first and fifth metatarsal heads, heels, lateral rear of feet, medial side of both feet, and triad marker plates were placed on the subjects' thighs and shanks. During static and dynamic trials, a vest was worn with reflective markers placed on the

shoulders, sternum, and upper back region. For dynamic trials, the following markers were removed: top head, lateral epicondyles of the humeri, styloid processes of the radii, iliac crests, ankle lateral and medial malleoli, first metatarsal heads, and medial sides of the feet.

Each subject performed five dynamic trials of walking, single leg squat (dominant leg/ previously injured leg), double leg squat, and drop jump from a 30 cm box. During landing trials, subjects were instructed to land as they normally would, then quickly explode into a jump. Subjects were also verbally instructed to not go past 90 degrees of knee flexion. During the squatting trials, the subject was verbally cued to do a 2:1:2-second squat tempo. That is 2 seconds on the descent, one second pause at the bottom, followed by a 2 second ascent. Subjects were not constrained to specific knee angles. Subjects used a self-selected pace for walking.

Data Reduction

Ultrasound Reduction

Ultrasound images were imported and analyzed using OsiriX DICOM Viewer software (Pixmeo, Bernex, Switzerland). Cross sectional area of the biceps femors long head was determined at each of the eleven slices using the closed polygon tool. (Figure 1A). The average of the two trials were determined and used for volume estimation. These cross sectional area measurements were used to estimate muscle volume integrating the area under the CSA vs muscle length curve. (Figure 2) Pennation angles and fascicle lengths were measured of the Vastus Lateralis (VL), Vastus Medialis (VM), Vastus Intermedius (VI), Rectus Femoris (RF), BFLH, BFSH, ST and SM.(Figure 1C) Three measurements of fascicle lengths were taken (distal, middle, and proximal) using the open polygon tool. These measurements were averaged together for analysis. Pennation angle measurements were taken at the end of each fascicle at the point where they inserted onto the aponeurotic tendon.

Figure 2: BFLH Cross Sectional Area vs. Muscle Length

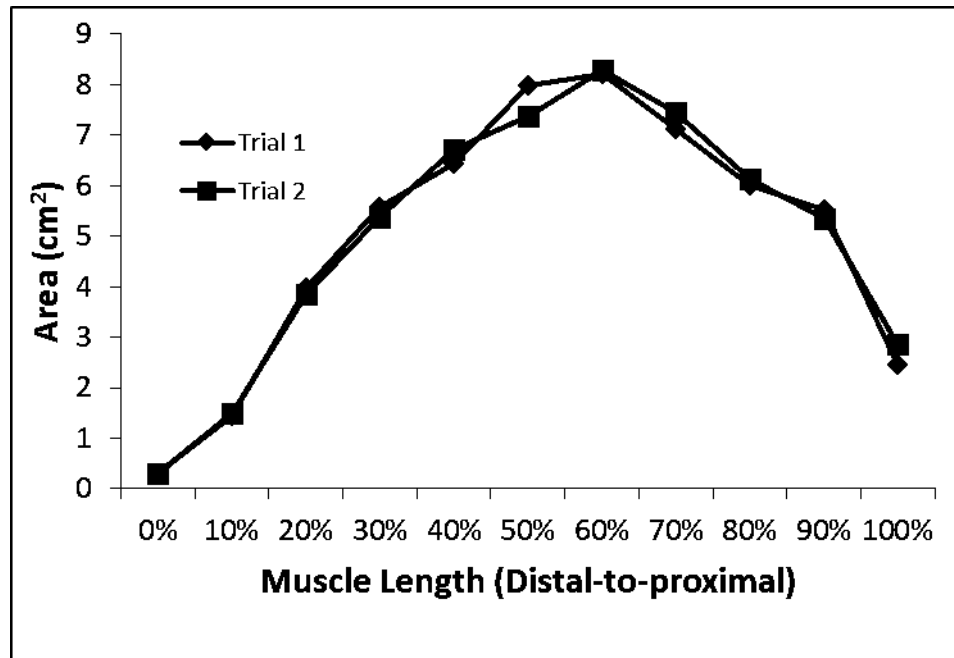


Figure 2: Biceps femoris long head area at each slice along the muscle length. 0% represents the smallest distal traceable area, 100% represents the smallest proximal traceable area at the gluteal fold. Images were taken twice at each slice for accuracy.

EMG and Motion Capture Data Reduction

Kinematic, kinetic, and EMG data were imported into Visual3D (CMotion Inc, Rockville, MD) where an 8 segment model was created. Kinematic data were low-pass filtered using a second order butterworth bidirectional filter at 6Hz. Kinetic force data were low-pass filtered at 50Hz. All surface EMG signals were high pass filtered at 30 Hz and full-wave rectified, then low pass filtered at 4 Hz. EMG signals were normalized to each subjects percent maximum voluntary contraction (%MVC)⁶⁶. A majority of the maximum EMG signal values used to normalize came from isometric dynamometer trials. However, some maximum signals occurred during dynamic trials. For example, the gastrocnemius muscles were commonly

normalized from the drop landing trials because of the maximal nature of the jumping motion. Vastus Lateralis EMG were often normalized from isometric trials on the dynamometer. Inverse dynamic calculations were performed in Visual 3D. All biomechanical measures were visualized on a report template for quality control.

Creating a Subject Specific Model

Using the ultrasound and static calibration data, a subject specific musculoskeletal model was created in Software for Interactive Musculoskeletal Modeling (SIMM)⁶⁷. The generic model used to create the subject specific model in SIMM had 25 degrees of freedom. 3 rotations about the lower torso, right and left sides include all of the following: arm adduction, arm rotation, arm flexion, elbow flexion, pronation/supination of forearm, hip adduction, hip rotation, hip flexion, knee flexion, ankle dorsi/plantar flexion, and subtalar inversion/eversion.

First, the model was scaled from a generic model⁶⁷ to match the subject's segment sizes. This was done from the static calibration motion capture trial, along with the subject's height and mass measurements. Wrapping objects were manipulated when necessary if unrealistic changes in musculotendon lengths were visible. This occurs when we scale the generic model. Wrapping errors occur when segments are scaled non-uniformly (XYZ dimensions) to the subjects segment size.

Subject-specific muscle force producing parameters were then derived from a combination of ultrasound and scaled SIMM musculotendon lengths. Maximal isometric force (F_{max}), optimal fiber length (FL_{opt}), pennation angle at optimal fiber length (θ_{opt}), and tendon slack length (TSL) were derived from ultrasound-based measurements and subject specific musculotendon lengths.

Subject-specific muscle model parameters were determined based on ultrasound data. Optimal fiber length was calculated by taking the fascicle lengths determined from ultrasound, and multiplied by the ratio of optimal sarcomere length to resting sarcomere length.⁶⁸ (Figure 4.1) Pennation angle at optimal fiber length was calculated using raw fascicle lengths and raw pennation angles as measured from ultrasound data. (Figure 4.2) F_{max} was calculated as the physiological cross sectional area multiplied by specific tension. Physiological cross sectional area was used as a calculation of muscle volume divided by the previously calculated optimal fiber length, times the cosine of the pennation angle at optimal fiber length. Specific tension is the force produced per unit of area, and was set at 35 N/cm².⁶⁹ (Figure 4.3) Tendon slack length is the length at which if the tendon were stretched any further, force would be produced.⁷⁰ To calculate tendon slack length, musculotendon lengths, optimal fiber length, pennation angle at optimal fiber length, and normalized fiber length (0.5-1.5) are needed. (Figure 4.4) Musculotendon lengths were determined from the scaled model. Tendon slack length was manipulated for biarticular muscles due to an unrealistic representation of fiber length with the hip flexed to 90 degrees and knee fully extended.⁷¹ Specifically, we manipulated TSL so that the normalized muscle fiber lengths did not exceed the normal physiological end range of 1.5 normalized fiber lengths. The ultrasound and model based muscle parameters for three of the seven muscles used in our model can be found in Appendix D.

Figure 3: Subject Specific Muscle Model Parameter Calculations

$$\begin{aligned}
 (1) \quad FL_{opt} &= FL_{raw} \left(\frac{S_{opt}}{S_{rest}} \right) & (3) \quad F_{max} &= \frac{Volume}{FL_{opt} * \cos(\theta_{opt})} * Specific\ Tension \\
 (2) \quad \theta_{opt} &= \sin^{-1} \left(\frac{FL_{raw} \sin \theta_{raw}}{FL_{opt}} \right) & (4) \quad TSL &= MT_{length} - \left(FL_{opt} * \sqrt{FL_{norm}^2 - \sin^2 \theta_{opt}} \right)
 \end{aligned}$$

Figure 3: Muscle model calculations

Estimation of Muscle Forces

Muscle forces were estimated using a hybrid static optimization procedure. C3D files from Visual 3d containing kinematic and kinetic data were imported into SIMM for each of the trials. Individual muscle forces were calculated using a modified Hill-type muscle model based on muscle tendon lengths and activation data.⁶⁶ The equation used to derive muscle forces at each point in time is a function of Fmax, which is modified by force-length, force velocity, parallel passive elastic force-length, activation level, and pennation angle (Figure 4).

During the optimization process, the original EMG signals were manipulated when calculating muscle forces. This static muscle optimization algorithm uses a least-squares equation to find activations that result in the muscle moments matching inverse dynamics moments. A confidence range is used to control for unreasonable EMG values. A correlation analysis was used to report r values between the experimental EMG and the optimized EMG for all four dynamic trials (Appendix: A). Correlations for the VL ranged from 0.67 to 0.99 for all dynamic tasks. Correlations for the BFLH ranged from 0.61 to 0.76 for all dynamic tasks. Correlations for the LG ranged from 0.21 to 0.95 for all dynamic tasks. The magnitudes changed during the optimization process, however we were confident in using these data to predict muscle forces because the overall pattern of the EMG signals stayed relatively the same.

The term muscle activation is used often in this document, however what is actually being measured by the EMG sensors is muscle excitation. Muscle excitation is the calcium ion concentration within the muscle, or cross bridge formations. True muscle activation is derived from the raw muscle excitation and a first order differential equation. This coupling between excitation and activation is used as an input into the muscle contraction dynamics.

Figure 4: Equation for Estimation of Muscle Forces

$$F^{mt} = F^{max} [f(l)f(v)a(t) + F_p(l)] \cos(\phi(t))$$

Figure 4: Muscle Force Equation. F^{mt} = Force produced by muscle tendon unit. F^{max} = maximum isometric muscle force. $f(l)$ = Hill type generic force-length. $f(v)$ = force velocity. $a(t)$ = muscle activation level. $F_p(l)$ = parallel passive elastic force-length. $\phi(t)$ = pennation angle.⁶⁶

When running the dynamics pipeline in SIMM, all data points were normalized to 100 points to be able to visualize and to compare the within and between group statistics. Event times were used from specific event detection pipelines that were ran in Visual 3D. Figure 5 shows an example of the muscles forces derived from the SIMM dynamics pipeline for each task for one subject.

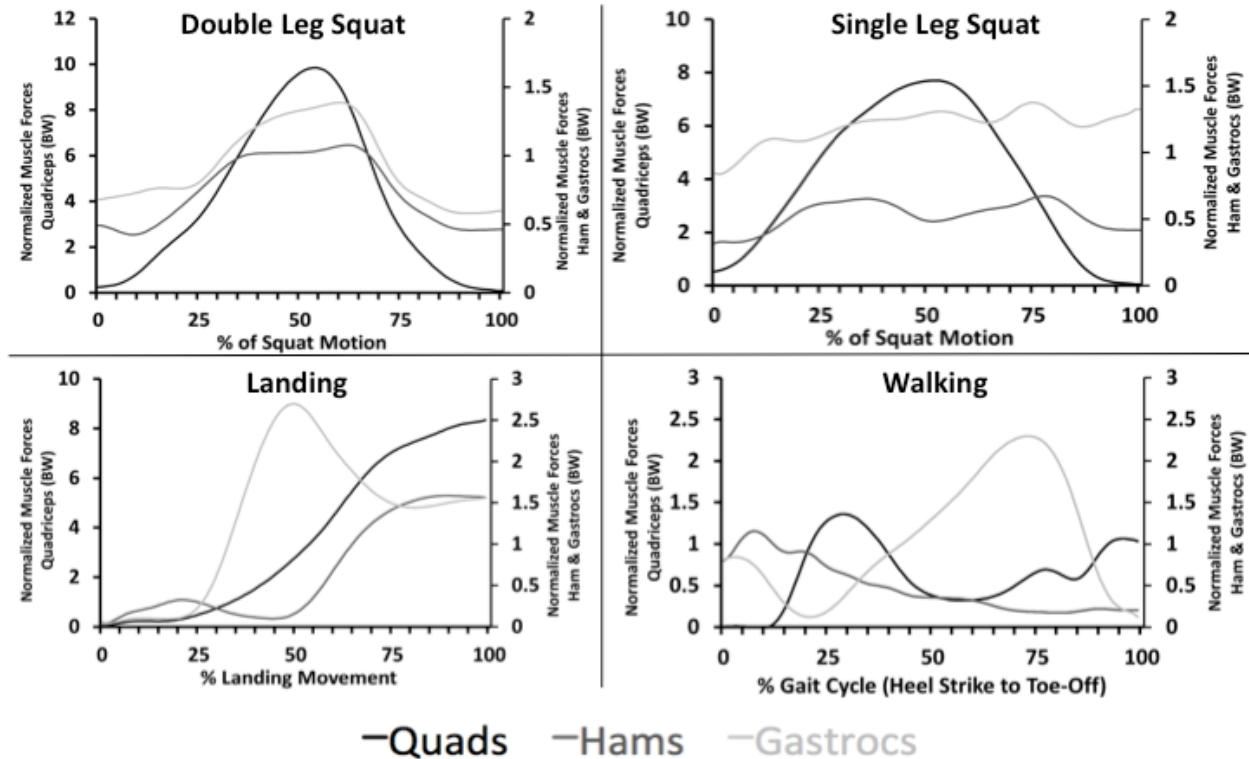
Figure 5: Muscle Forces for each Dynamic Task

Figure 5: Example of muscle forces during each dynamic task (one subject). Quadriceps (Quads) forces are black, Hamstrings (Hams) are dark grey, and Gastrocnemius (Gastrocs) are light grey. All muscle forces were normalized to the subjects body weight in newtons. Ground contact during landing occurred at 25% of the movement, 25%-100% indicate 150ms after ground contact. Walking figure shows heel strike (0%)

until toe off (100%). The Primary vertical axis (left) represents the Quadriceps forces and the secondary vertical axis (right) represents the hamstrings and gastrocnemious forces.

Estimation of ACL Forces

An ACL model⁷² was used to compute ACL forces. Muscle forces (quadriceps hamstring, and gastrocnemius) and joint reaction forces were resolved into shear and compressive components relative to the tibia, which also accounts for a posterior tibial slope of 7 degrees.⁷³ A polynomial function was then used to determine the magnitude of shear forces imparted on the ACL based on the elevation angle relative to the surface of the tibia as a function of knee angle during weight bearing activities.⁷⁴ Figure 6 shows an example of ACL forces during each dynamic trial.

ACL force patterns are reasonable based on previous literature.^{75,76} Similar to our ACL forces in walking, Shelburne et al. (2004) showed ACL forces that follow a similar bimodal trend.⁷⁵ Escamilla et al. (2008) reported ACL forces using a static optimization musculoskeletal modeling technique during a double leg squat. Our ACL forces during double leg squat are reasonable based on the pattern shown by Escamilla et al.⁷⁶ In landing, Pflum et al. (2004) reported peak ACL forces near the time of peak vertical ground reaction force, similar to what our model predicts.⁶³

Strain on the ACL has been measured during squatting tasks. Beynnon et al. (1997) found that strain decreases as a function of increased knee flexion angle. Specifically, the ACL exhibits between 3-4% strain during extension, and is completely unloaded (below 0%) between 40-100 degrees.⁷⁷ Cerulli et al. (2003) found that ACL strain increased by 5% at the time of peak vertical ground reaction force after landing from a single leg hop.⁶⁰

Figure 6: ACL forces during each dynamic task

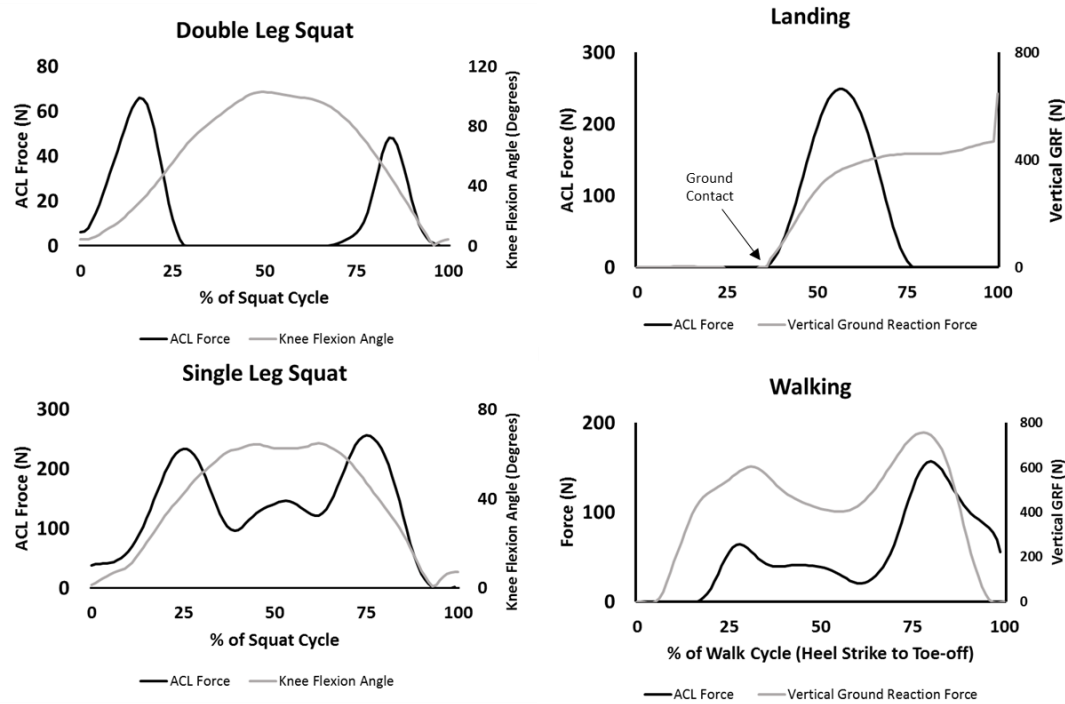


Figure 6: ACL forces during each dynamic task. Squatting trials show ACL force (black) and knee flexion angle (grey). Landing and walking show ACL force (black) and vertical ground reaction force (grey).

In summary, the subject specific model was created by scaling to segment sizes based on static calibration data from motion capture, then ultrasound measurements were used to predict muscle force producing parameters. Muscle and joint reaction forces, along with kinematics, were then be used to estimate ACL forces.⁷²

Data Analysis

We tested the hypothesis that people with prior hamstring strain injuries have higher ACL forces. Squatting used a 2 (group: healthy vs previous hamstring injury) X 14 (Knee Angle: 7 descent bins and 7 ascent bins) repeated measures ANOVA. Walking used a 2 (group) X 2

(ACL force peaks) repeated measures ANOVA. Landing used an independent samples t-test to show the relationship between groups and peak ACL force. Statistical significance was set at $P < 0.05$.

Chapter 4: Results

Results for Primary Hypothesis

We hypothesized that people with a previous hamstring strain injury who were otherwise healthy at the time of data collection would have increased forces on their ACL compared to health individuals. Of the four dynamic tasks, landing had the highest ACL forces, followed by single leg squatting, then walking, and double leg squat had the least ACL forces.

Table 2: ACL Forces Between Groups in Each Dynamic Task

| | Double Leg Squat | Single Leg Squat | Landing | Walking |
|---------------------------------------|-------------------|-------------------|-------------------|------------------|
| Control | 0.03 ± 0.03 | 0.24 ± 0.09 | 0.56 ± 0.21 | 0.19 ± 0.08 |
| Knee Angle @ Peak ACL Force (Degrees) | 29.86 ± 7.22 | 37.03 ± 18.85 | 33.12 ± 12.05 | 10.73 ± 3.39 |
| Previous HSI | 0.02 ± 0.03 | 0.19 ± 0.13 | 0.58 ± 0.33 | 0.20 ± 0.12 |
| Knee Angle @ Peak ACL Force (Degrees) | 32.59 ± 25.38 | 41.97 ± 3.07 | 44.83 ± 6.46 | 13.70 ± 1.15 |

Table 2: Between-group ACL forces normalized to each subjects body weight (N). Squatting trials represent the mean ACL forces across the entire trial. Landing ACL forces were calculated as the peak value for each subject occurring after ground contact. Walking ACL forces were calculated as the average of the two peaks for each subject. Knee angles at peak ACL forces (degrees) were calculated as a five-trial average for each subject during each dynamic task. No significant differences between groups were found for any of the dynamic tasks.

Double Leg Squat

A 2 (group) x14 (knee angle) repeated measures ANOVA compared ACL forces in increments of 10 degrees of knee flexion angle (starting at 15°) throughout the double leg squat cycle. The within-group effect for knee angle was statistically significant ($p<.01$) showing the ACL forces changed as a function of knee flexion angle during descent and ascent phases of the squat. The ACL forces were significantly higher at knee angles closer to extension compared to the most flexed angles (Figure 7, Descent 15-55degrees). ACL forces were statistically different

than the bottom 4 angle bins (Descent 65-80 and Ascent 65-80) $p < .05$, and Ascent (15-45 degrees) ACL forces were statistically different than the bottom 2 angle bins (Descent 75-80 and Ascent 75-80) $p < .05$). While the ACL forces appeared higher during descent compared to ascent at lower flexion angles (15-55 degrees), these values were not statistically different (15-25 degrees, $p = .056$, 25-35 degrees, $p = .087$, 35-45 degrees, $p = .170$, 45-55 degrees, $p = .317$). There was no statistically significant difference between healthy vs subjects with previous history of hamstring strain (Figure 8, $p = .227$). The control group mean normalized ACL force during double leg squat was 0.028 ± 0.03 BW while the hamstring injury group was 0.019 ± 0.03 BW.

Figure 7: Double Leg Squat Normalized ACL Force by Knee Angle Control vs. Previous HSI

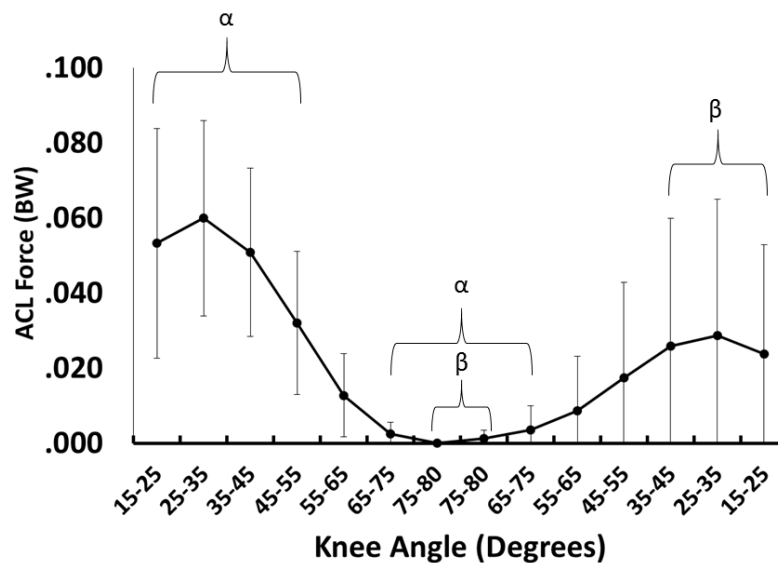


Figure 7: ACL Forces Normalized to Body Weight (BW). α = Descent (15-55degrees) ACL force is statistically different then bottom 4 angle bins (Descent 65-80 and Ascent 65-80) $p < .05$. β = Ascent (15-45 degrees) ACL force is statistically different then bottom 2 angle bins (Descent 75-80 and Ascent 75-80) $p < .05$.

Figure 8: Double Leg Squat Normalized ACL Forces – Control vs. Previous HSI

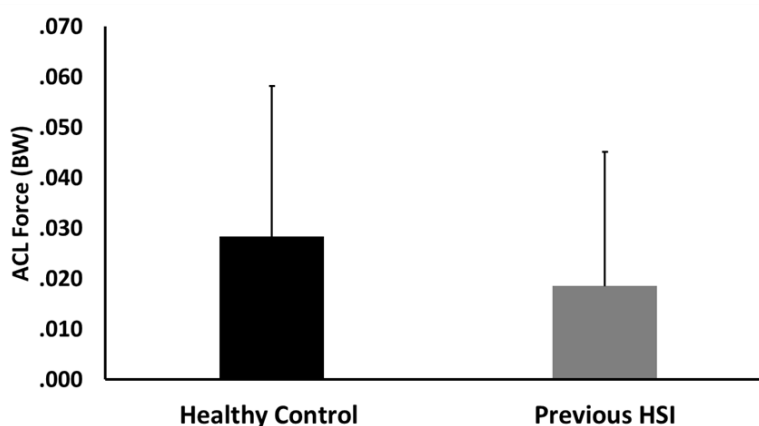


Figure 8: ACL Forces Normalized to Body Weight (BW). HSI is Hamstring Strain Injury. ($p = .227$)

Single Leg Squat

The within-subjects comparison of ACL forces based on knee angle was statistically significant (Figure 9, $p < .01$). The ACL forces were significantly higher at knee angles closer to extension compared to the most flexed angles (Figure 9, Descent (25-55 degrees) ACL forces were statistically different than the bottom 4 angle bins (Descent 65-80 and Ascent 65-80) $p < .05$, and Ascent (35-55 degrees) ACL forces were statistically different than the bottom 4 angle bins (Descent 65-80 and Ascent 65-80) $p < .05$). Between groups comparison showed no statistical difference (Figure 10, $p = .487$). The healthy control group mean normalized ACL force during single leg squat was 0.242 ± 0.09 BW while the hamstring injury group was 0.194 ± 0.13 BW.

Figure 9: Single Leg Squat Normalized ACL Force by Knee Angle Control vs. Previous HSI

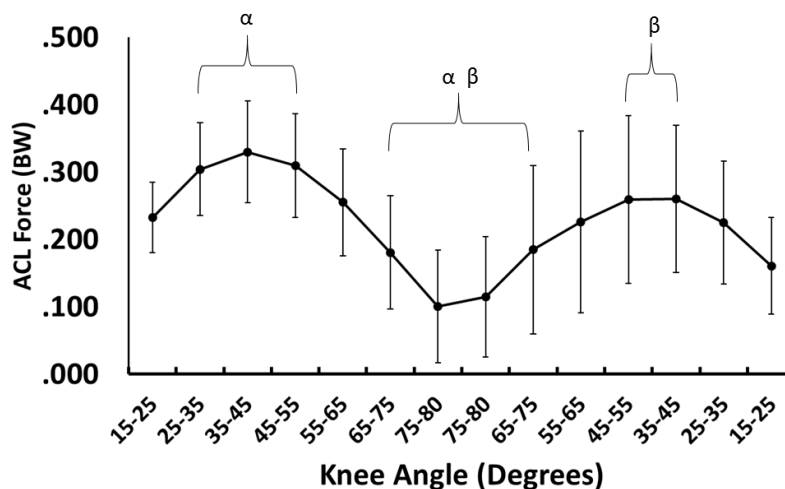


Figure 9: ACL Forces Normalized to Body Weight (BW).
 α = Descent (25-55degrees) ACL force is statistically different then bottom 4 angle bins (Descent 65-80 and Ascent 65-80) $p < .05$.
 β = Ascent (35-55 degrees) ACL force is statistically different then bottom 4 angle bins (Descent 65-80 and Ascent 65-80) $p < .05$.

Figure 10: Single Leg Squat Normalized ACL Forces – Control vs. Previous HSI

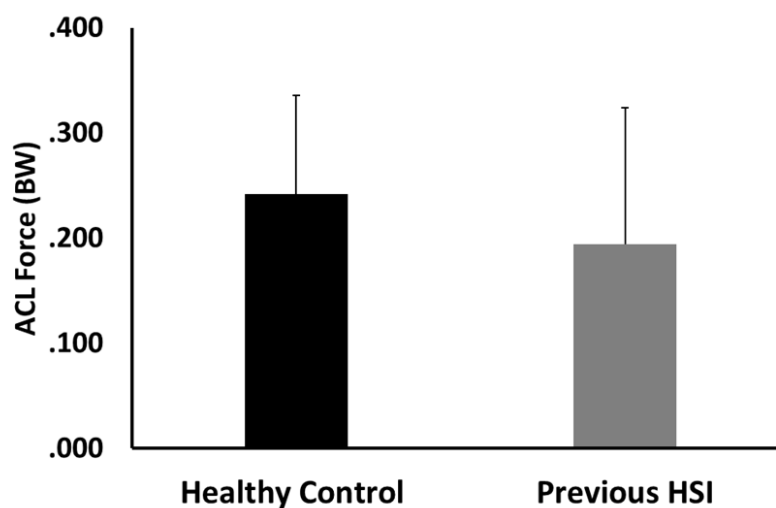


Figure 10: ACL Forces Normalized to Body Weight (BW). HSI is Hamstring Strain Injury. ($p = .487$)

Landing

The between group ACL forces were not statistically significant ($p = .389$). The mean peak normalized ACL forces for the healthy control group were 0.56 ± 0.21 [0.39, 0.73] BW, and for the previous HSI group it was 0.58 ± 0.33 [0.12, 1.04] BW. (Figure 11).

Figure 11: Peak Normalized ACL Forces in Landing Control vs. Previous HSI

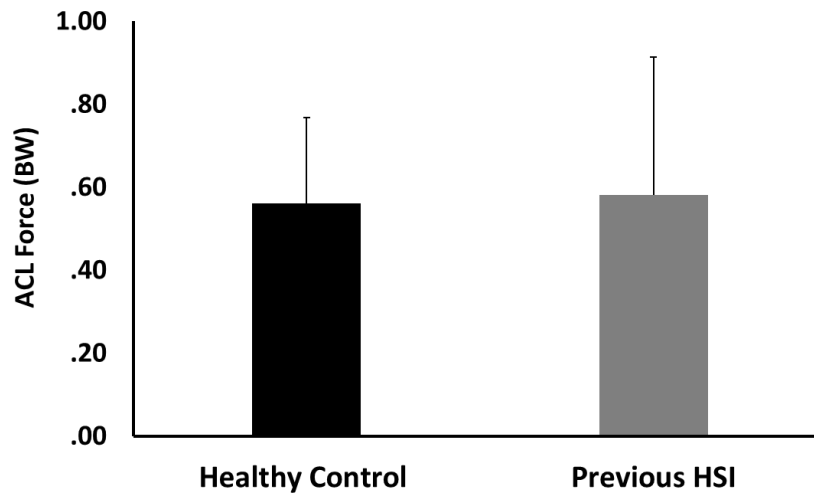


Figure 11: ACL Forces Normalized to Body Weight (BW). HSI is Hamstring Strain Injury. ($p = .389$)

Walking

Between groups comparison showed no statistical difference (Figure 12, $p = .826$). The within-subjects comparison of ACL forces based on first vs second peak was not statistically different (Figure 13, $p = .101$).

Figure 12: Normalized Peak ACL Forces in Walking Control vs. Previous HSI

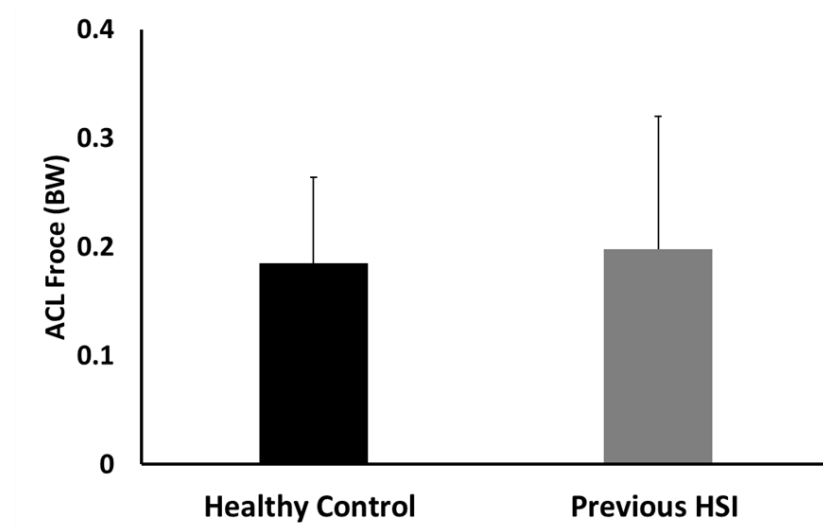


Figure 12: ACL Forces Normalized to Body Weight (BW). HSI is Hamstring Strain Injury. ($p = .826$)

Figure 13: Two Peak Comparison - Normalized ACL Forces in Walking 1st Peak vs. 2nd Peak

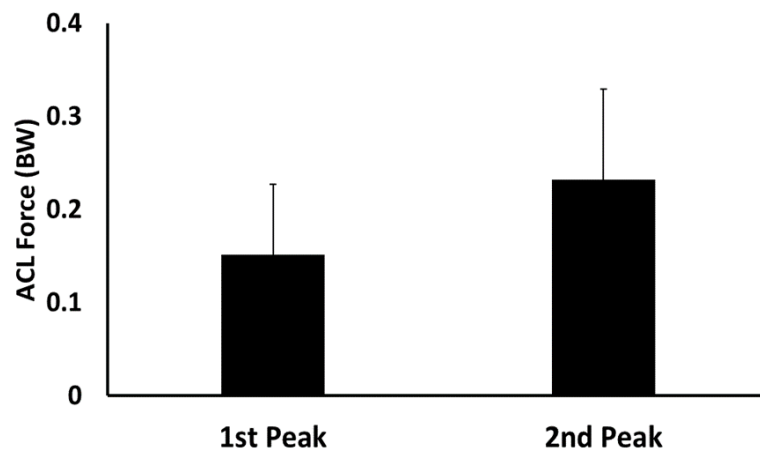


Figure 13: ACL Forces Normalized to Body Weight (BW). Walking produced two separate peaks for ACL forces from heel strike to toe off. ($p = .101$)

Summary of Results

Overall, there were no significant differences in ACL forces between groups for all four dynamic tasks. During the squatting tasks, ACL forces were generally higher at lower knee flexion angles near extension.

Chapter 5: Discussion

The purpose of this study was to determine if people with a previous hamstring strain had increased ACL forces during common activities of daily living and sport compared to healthy controls. To test this, we used an ultrasound based subject-specific musculoskeletal model. The overall results did not support our hypothesis. The between-groups (healthy control vs. previous hamstring injury) comparisons indicated no statistical significance in ACL forces for all four dynamic tasks (double leg squat, single leg squat, landing, and walking). We speculate a few reasons to why the data did not support our hypothesis.

Do people with previous HSI move differently?

One plausible explanation for why ACL forces were not higher in the experimental group is that people with previous HSI move in such a way to minimize ACL forces in the previously injured limb. When calculating total sagittal plane range of motion (ROM) at the hip, knee, and ankle during all dynamic tasks, we found significant differences between the control group and previous HSI group (Table 2, Figures 14-17). During double leg squat control subjects had ROM at the hip of 100.9 ± 6.8 , while previous HSI subjects had 81.5 ± 8.4 (Figure 14, $p < .01$). Although not statistically significant, ROM at the knee during double leg squat for controls was 101.2 ± 6.1 , and for previous HSI it was 90.5 ± 9.7 ($p = .063$) No notable differences were seen at the ankle for double leg squat (Table 2).

**Table 3: Sagittal Plane Range of Motion
Previous HSI vs. Healthy Control**

| Task | | Previous HSI | Control | p-value |
|-----------|-------|--------------|-------------|---------|
| DLS | Hip | 81.5 ± 8.4 | 100.0 ± 6.9 | 0.01* |
| | Knee | 90.5 ± 9.7 | 101.2 ± 6.1 | 0.06 |
| | Ankle | 22.9 ± 3.5 | 23.3 ± 3.9 | 0.89 |
| SLS | Hip | 64.1 ± 5.3 | 84.4 ± 7.8 | 0.01* |
| | Knee | 67.8 ± 6.6 | 71.3 ± 9.5 | 0.59 |
| | Ankle | 27.2 ± 3.9 | 25.3 ± 3.5 | 0.48 |
| Drop Jump | Hip | 59.5 ± 11.9 | 75.1 ± 11.5 | 0.07 |
| | Knee | 82.3 ± 3.7 | 90.0 ± 7.9 | 0.16 |
| | Ankle | 58.3 ± 13.6 | 68.6 ± 8.4 | 0.17 |
| Walking | Hip | 33.7 ± 2.4 | 42.0 ± 5.6 | 0.05* |
| | Knee | 41.1 ± 7.8 | 38.6 ± 2.3 | 0.10 |
| | Ankle | 26.5 ± 3.4 | 30.0 ± 5.5 | 0.36 |

Table 2: Sagittal plane range of motion (degrees) between previous HSI group and healthy control group. DLS is double-leg squat task and SLS is single-leg squat task. * Represents statistically significant at the $p < .05$ level.

**Figure 13: Angular Position of Hip, Knee, and Ankle during
Double Leg Squat**

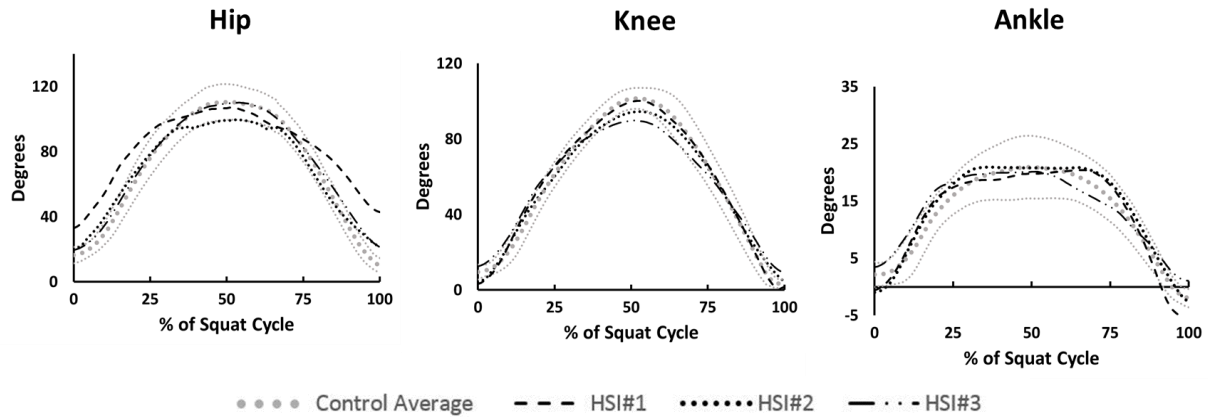


Figure 13: Sagittal Plane Angular Positions during Double Leg Squat. Large grey circles indicate the average of the control subjects, the small grey circles are \pm the standard deviations. The individual black lines are the three individual previous HSI subjects. ROM at each joint was calculated as the difference between the maximum flexion angle and the minimum flexion angle on average for all five dynamic trials.

Single leg squat ROM control subjects averaged $83.2 \text{ degrees} \pm 7.8$ at the hip, while previous HSI subjects had 64.1 ± 5.3 (Figure 15, $p < .01$). No notable differences were seen at the knee or ankle for single leg squat (Table 2).

Figure 14: Angular Position of Hip, Knee, and Ankle during Single Leg Squat

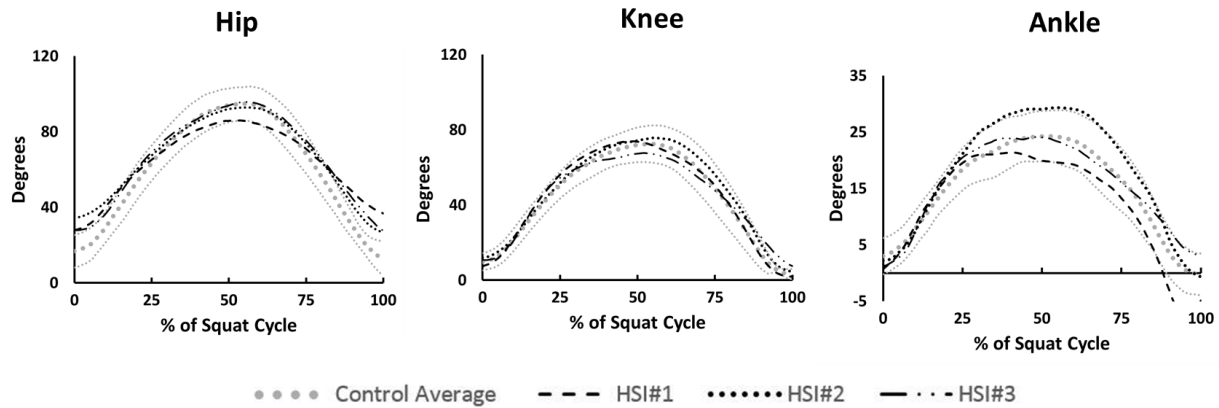


Figure 14: Sagittal Plane Angular Positions during Single Leg Squat. Large grey circles indicate the average of the control subjects, the small grey circles are \pm the standard deviations. The individual black lines are the three individual previous HSI subjects. ROM at each joint was calculated as the difference between the maximum flexion angle and the minimum flexion angle on average for all five dynamic trials.

During the drop jump, although not statistically significant, control subjects had mean ROM at the hip of $75.6 \text{ degrees} \pm 10.5$, while previous HSI subjects had 59.5 ± 11.9 (Figure 16, $p = .065$). No notable differences were seen at the knee or ankle for the drop jump task (Table 2).

Figure 15: Angular Position of Hip, Knee, and Ankle during Drop Jump

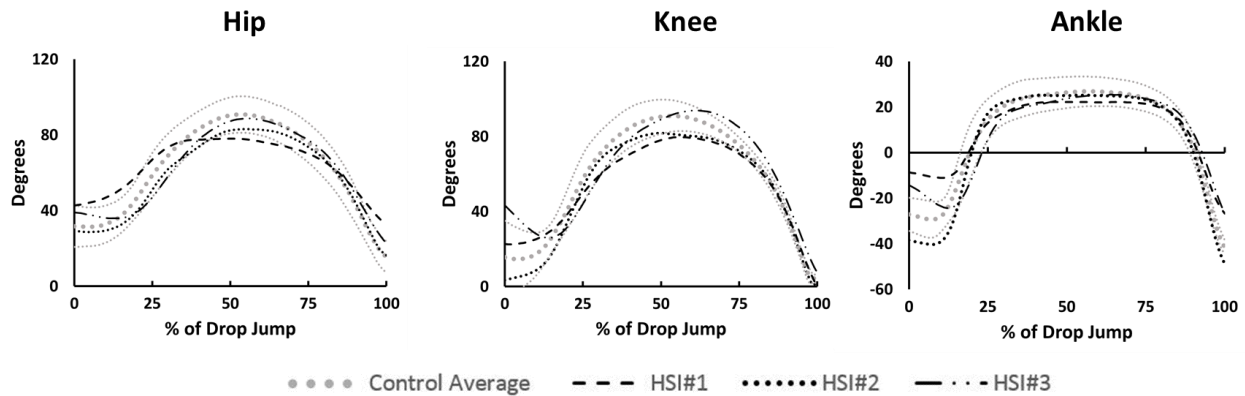


Figure 15: Sagittal Plane Angular Positions during Drop Jump Task. 0% of drop jump represents just prior to ground contact from landing, and 100% represents toe-off as they launch into the air. Large grey circles indicate the average of the control subjects, the small grey circles are \pm the standard deviations. The individual black lines are the three individual previous HSI subjects. ROM at each joint was calculated as the difference between the maximum flexion angle and the minimum flexion angle on average for all five dynamic trials.

Walking trials showed a significant difference in Hip ROM between groups, controls displayed a mean ROM of 41.4 ± 5.4 , while previous HSI showed a mean ROM of 33.7 ± 2.4 (Figure 17, $p < .05$). No notable differences were seen at the knee or ankle for walking (Table 2).

Figure 16: Angular Position of Hip, Knee, and Ankle during Walking

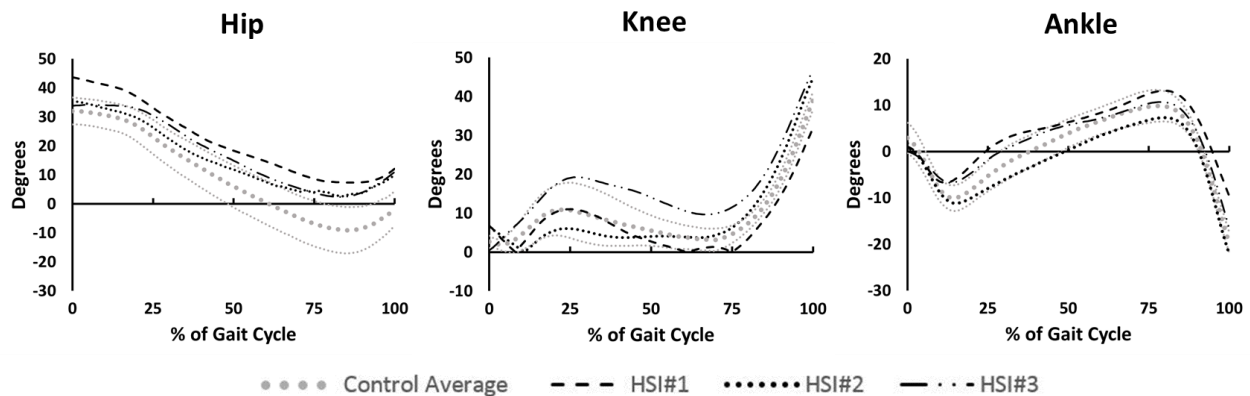


Figure 16: Sagittal Plane Angular Positions during Drop Jump Task. % of Gait Cycle represents heel strike to toe off. Large grey circles indicate the average of the control subjects, the small grey circles are \pm the standard deviations. The individual black lines are the three individual previous HSI subjects. ROM at each joint was calculated as the difference between the maximum flexion angle and the minimum flexion angle on average for all five dynamic trials.

Collectively, it appears that our HSI subjects accomplished the tasks with overall less range of motion, compared to the healthy controls. These kinematic differences may have developed to restrain ROM of the hip and knee such to avoid potentially dangerous strains on ligamentous structures of the lower extremity. Although speculative, it could explain why we did not see differences in ACL forces.

Movement adaptations post-injury are supported in the literature, however there is little evidence to support that people move differently after hamstring strain injury. One study by Brughelli et al. (2010) showed differences in the horizontal ground reaction forces during sprinting between previous hamstring injured limb and the contralateral healthy limb.⁷⁸ Although this study mostly compared asymmetries between limbs, there was also a difference in horizontal force between the injured limb in the injured group and the bilateral average of the non-injured group. Another study by Brockett et al. (2004) showed that people with a history of hamstring injury produced peak knee flexor torque at a higher knee flexion angle compared to healthy control subjects (53.5° in previous HSI group and 37.5° in healthy control group).⁷⁹ Since peak torque is occurring at a shorter muscle length, they concluded that this could be a post-injury mechanism by protecting the hamstring musculotendon unit to limit strain on the muscle and ultimately decrease injury risk. It is worth noting (although not statistically significant) that during our isokinetic trials that subjects with previous HSI produced peak knee flexor torque at a higher knee flexion angle 35.7 ± 6.1 degrees, compared to our healthy control group 27.8 ± 11.5 degrees ($p = .145$).

To support the between group differences in ROM after injury, we explored previous literature to provide evidence that movement kinematics are different between groups in people with ligamentous and joint injuries vs. healthy individuals.

Differences in kinematics and kinetics after injuries to the ligaments and joint structures of the lower extremity have been well documented in the literature. Chances of getting knee osteoarthritis (OA) have been shown to increase in people with previous ACL reconstruction (ACLR) surgery.³ In walking, Kaufman et al. (2001) found that people with knee OA have significantly decreased internal knee extensor moments and concluded that people with knee OA adapt their gait characteristics to move in such a way to decrease knee joint loading.⁸⁷ Ultimately, these changes in gait are likely a mechanism for decreasing pain in the osteoarthritic knee. Whether the person is voluntarily or involuntarily moving in that way, it appears that an altered movement strategy has been adapted.

ACL deficient (ACLD) and ACLr populations have shown to have different kinematics during many different dynamic movements after injury or surgery compared to healthy individuals. Decker et al. (2002) showed that people with previous ACLr utilized an adapted landing strategy compared to healthy subjects. Specifically, ACLr subjects demonstrated a more erect landing posture, which in turn lead to less hip extensor muscles and more ankle plantarflexors muscles being used. The authors suggested that the ACLr group adopted a compensation strategy in landing to avoid the hip extensor muscles.⁸⁸ Yamazachi et al. (2009) found differences in kinematics in ACLd subjects during single leg squat compared to healthy controls. In male subjects, the ACLd group had significantly less external rotation of the hip at peak knee flexion compared to male controls. In female subjects, the ACLd group showed more hip external rotation and knee flexion, and less hip flexion compared to female control subjects.⁸⁹ After ACL injury, no matter if someone decides to have or not have reconstructive surgery, their kinematics have been shown to be different than people with intact ACLs. Findings by both

Decker et al. and Yamazachi et al. support an alternative hypothesis that people move differently after injury. In both of these studies, and our current study, hip ROM was significantly less.

There are potentially many different injuries (acute and chronic) that can change the way someone moves. It is not clear though if these adaptations are beneficial or harmful to the individual. Since most of these studies (including ours) are retrospective, it is hard to conclude that injury directly causes changes to their kinematics. However, we can speculate that these individuals with a previous acute or ongoing chronic injury may change the way they move to potentially decrease their risk of another injury. In our study, we found significant decreases in hip flexion in three of the four tasks. Decreased hip flexion may interfere with hamstrings protective role by shortening the hamstrings and reducing its force, and thereby potentially increasing ACL forces.

Muscle Activations after HSI

Differences in muscle activation patterns post hamstring injury have been well documented.^{16,17,50,51,90} For two of the previous HSI subjects we saw an increase in lateral hamstring EMG during the impact phase (ground contact to zero vertical velocity - just before acceleration upward into jump) of the drop jump. Compared to the control mean \pm standard deviation activations, several deviations of activation fell above or below 1SD for the HSI subjects. For the lateral hamstrings (LH), previous HSI subject 1 displayed maximum peak EMG (%MVC) during the impact phase of 33.7% (Figure 18, arrow A), while the control average was $12.7 \pm 12.0\%$ at the same point in time after ground contact. HSI subject 2 displayed a maximum peak EMG (%MVC) during the impact phase of 37.0% (Figure 18, arrow B) for the lateral hamstrings. All three previous hamstring subjects had increased medial hamstring (MH) peak EMG (%MVC) during the impact phase of a drop landing (Figure 18, arrow C). The

previous HSI group had average peak EMG during the impact phase of $15 \pm 1\%$, and the control group was $7 \pm 2\%$ (Figure 18). We speculate that this increase in hamstring activation during the impact phase of a drop jump could be a protective mechanism to prevent further injury. It is also worth noting that these increased EMGs are likely later compared to when the ACL forces peak (~time of peak vertical ground reaction forces, vertical line figure 17). This may be a delayed adaptive strategy and may not protect the ACL in high impact movements such as landing from a jump.

Figure 17: EMG of Four Lower Extremity Muscles during Drop Jump Task

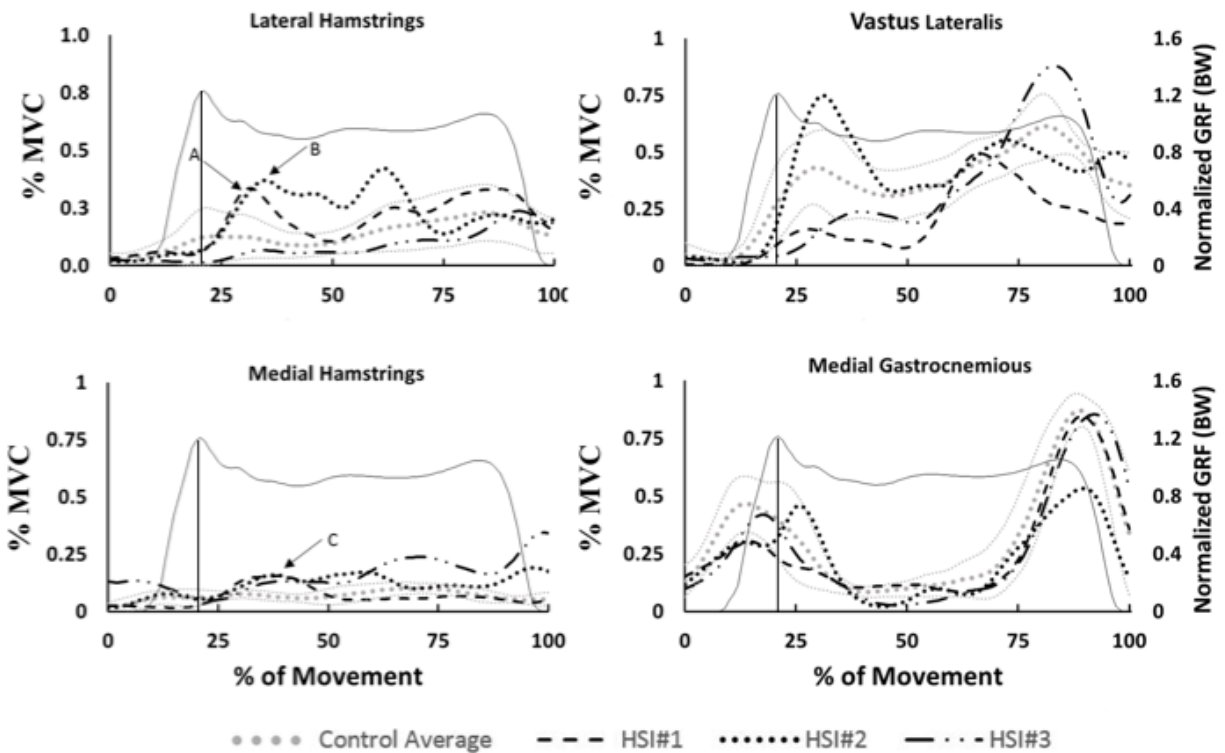


Figure 17: Electromyography (EMG) signals during drop jump task. All signals were processed and normalized to the individual subjects' maximum voluntary contraction (%MVC) voltage. Large grey circles indicate the average of the control subjects, the small grey circles are \pm the standard deviations. The individual black lines are the three individual previous HSI subjects. Solid grey line is the average normalized vertical ground reaction force during drop jump task. Vertical black lines indicate peak ground reaction force, which is associated with the time of peak ACL forces.

Table 4: Normalized (BW) Muscle Forces at Peak ACL Forces Between Groups

| Task | | Previous HSI | Control |
|----------------|----------|--------------|-------------|
| DLS | Quads | 1.60 ± 1.24 | 1.31 ± 0.22 |
| | Hams | 0.37 ± 0.19 | 0.41 ± 0.07 |
| | Gastrocs | 0.70 ± 0.21 | 0.77 ± 0.28 |
| SLS | Quads | 2.66 ± 1.60 | 3.61 ± 0.84 |
| | Hams | 0.32 ± 0.07 | 0.32 ± 0.11 |
| | Gastrocs | 1.30 ± 0.23 | 1.54 ± 1.15 |
| Landing | Quads | 3.11 ± 1.75 | 3.27 ± 1.22 |
| | Hams | 0.09 ± 0.01 | 0.23 ± 0.20 |
| | Gastrocs | 1.44 ± 1.23 | 2.53 ± 1.15 |
| Walking | Quads | 1.07 ± 0.47 | 0.43 ± 0.25 |
| | Hams | 0.30 ± 0.08 | 0.15 ± 0.08 |
| | Gastrocs | 1.37 ± 0.90 | 1.69 ± 1.00 |

Table 4: Muscle forces at peak ACL force normalized to body weight (BW).

During the drop jump trials, at the time of peak vertical ground reaction force (around the time of peak ACL force) HSI subjects had generally lower hamstring EMG. When looking at the muscle forces at peak ACL forces (Table 4), there is a noticeable difference between groups in hamstring muscle forces at peak ACL force. Specifically, the previous HSI subjects had 0.09 body weights of hamstring muscle forces, and the control group had 0.23 body weights hamstring force. One would expect that ACL forces would be significantly greater in this case since there is very little supplementary protection from the hamstrings to protect the ACL in the previously injured group. However, peak ACL forces in the previous HSI group occurred at a somewhat higher knee flexion angle compared to the control group (table 2). Since the

magnitude of ACL forces in the model relies on knee flexion angle, this could explain why ACL forces were not considerably higher in the previous HSI group.

Although we didn't see differences in ACL forces between groups, lower hamstring muscle activation has been related to ACL injury. In a study on females who later tore their ACL, all had lower pre-activity EMG of the hamstrings and increased pre-activity of the quadriceps.³⁴ Pre-activity of the hamstrings and quadriceps in our study, although higher for subject 3 in the medial hamstring, showed no differences. In the three HSI subjects, at around the time of peak ACL forces (40-60ms after ground contact), the lateral hamstrings were visibly less than the control averages ± 1 SD, suggesting that there would be a subsequent increase in ACL forces compared to the control group. However the amplitude of quadriceps activations were also lower than the control average, which could explain why we did not see a higher ACL forces in the HSI vs control groups.

Previous HSI Group was Stronger

As stated earlier, we found that the individuals in the HSI group did in fact move differently based primarily on the less total hip ROM. Muscle stiffness is one factor that has been shown to decrease passive ROM of the hamstrings for people with previous HSI.^{91,92} Blackburn et al. (2013) found that greater hamstring stiffness accounted for less ACL loading and injury risk during landing.⁹³ It is worth noting that our previous HSI males on average had higher passive torque generated by the hamstrings (33.16 ± 1.05 N*m) compared to our male control subjects (31.75 ± 8.42 N*m). This is not a large difference, however it could help to explain part of why we saw no differences in ACL forces. Also noteworthy, on average our previous HSI group had larger normalized (to body mass) BFLH volume (3.22 ± 1.00 cm³/kg) compared to controls (2.58 ± 0.41 cm³/kg). Related to that, not surprisingly we found that the total normalized

isometric and isokinetic hamstring torques were higher in the previous HSI group (1.89 ± 0.46 N*m/kg) compared to the control group (1.71 ± 0.45 N*m/kg). Previous HSI subjects were stronger, therefore this could have potentially had an impact in providing higher supplementary protection on the ACL during the dynamic tasks with generally lower EMG at the time of peak ACL forces.

We believe that an important limitation to mention is that the subjects that sustained a previous hamstring strain injury did not have functional deficits based on the hamstring function questionnaire values (table 1). All of our subjects had hamstring function scores over 80%. This may indicate that their hamstring healed well, and therefore had little/no functional deficits. If this is in fact the case, it could explain why we did not see any differences in ACL forces. Future research could include/exclude based on hamstring function questionnaire values to ensure that the individuals with previous HSI do in fact have functional deficits, and are potentially at high risk of injury.

Limitations and Future Research

Clearly sample size is one of the main limitations to this study. Based on the subject-by-subject comparisons of kinematics and muscle activations, future research could help to strengthen the argument that people move differently after HSI injury.

Prior to data collection, we had hoped to see the presence of scar tissue at the site of injury on the ultrasound images. We did not see any visible structural damage for any of the three hamstring injury subjects. Further research could use the presence of scar tissue as inclusion criteria. A couple other limitations to mention are the injury site and severity of injury. The location of the injury could potentially play an important role in the magnitude of the functional deficits after injury. Since we did not use injury site as inclusion/exclusion criteria,

this could be a limitation to why we did not see differences in ACL forces. Similarly, the severity of the injury could be important, and we limited our study to grade one and two hamstring strains. Future research could focus on just grade two hamstring strains, for example.

There are associated limitations whenever using a musculoskeletal model. Our static optimization method was used to predict muscle forces based on subject-specific activation data. These physiological measures help to generate a reasonable representations of muscle forces during dynamic tasks, however muscle forces are estimations and are not direct measurements. Another associated limitation to our model is the manipulation of EMG data during the optimization process. We were comfortable using it for our model because the activation patterns stayed relatively the same, however the magnitudes of the EMG used to predict muscle forces were not the same as the magnitudes of the experimental EMG signals.

Many studies that measure ACL forces during dynamic tasks are unanticipated in order to closely mimic in-game situations in which ACL injury often occurs. Our study used anticipated tasks which may have altered muscle excitation signals, and subsequently muscle and ACL forces. Future studies could have a design that includes both anticipated and unanticipated tasks to be able to see differences in muscle excitation signals between previously injured and healthy groups.

Conclusion

We did not support our original hypothesis that people with a previous hamstring strain injury would have increased ACL forces compared to healthy controls. However we proposed an alternative hypothesis that people move differently to minimize the risk of further injury. Range

of motion was different between groups for all four tasks. Specifically, the previous HSI group showed significantly less range of motion at the hip compared to the healthy control group in three of four dynamic tasks. EMG was qualitatively different during our highest effort movement, landing/drop jump. Since most ACL injuries occur during high impact movements such as landing from a jump, these data may be valuable to show neuromuscular adaptations after injury. Our previous HSI group was generally stronger than the control group; therefore future studies should focus on having matched strength between groups.

Future research should have more specific inclusion/exclusion criteria for determining people for the previously injured group. For example, one could limit the study to just people who previously sustained a grade two-hamstring strain. Doctor's records could also be helpful for including/excluding potential participants. Ultimately, having more specific inclusion criteria would help to support the new hypothesis that people with a history of hamstring injury move differently. Although, since this experimental design was retrospective, we cannot conclude that these movement adaptations were a result or cause of injury. However we speculate that substantial changes to the hamstrings could alter someone's kinematics to decrease their risk of further injury. If future studies found this to be true, it is a logical explanation for why we did not see differences in ACL forces.

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Appendix A: Figure 19 - EMG Correlation Analysis

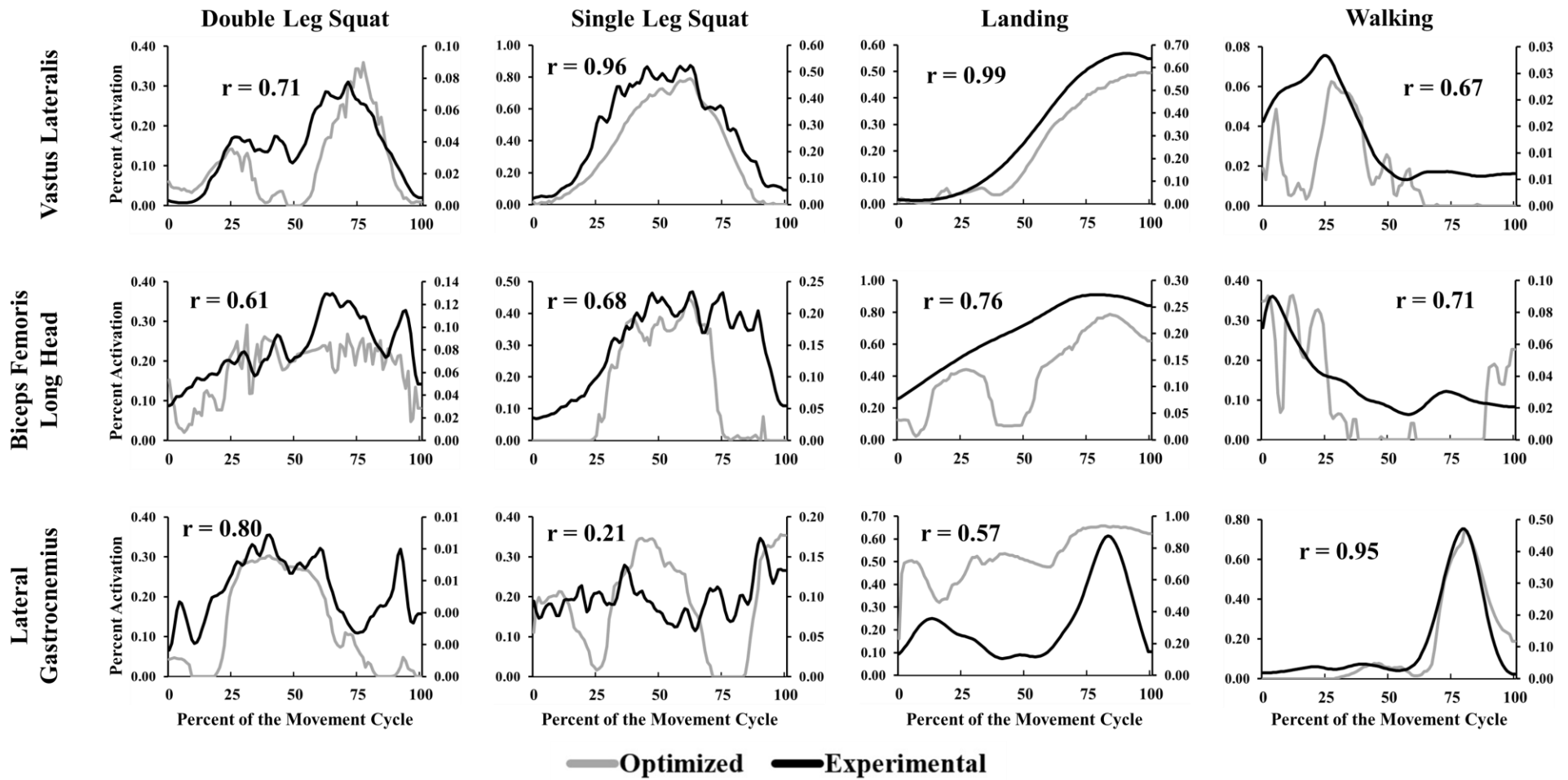
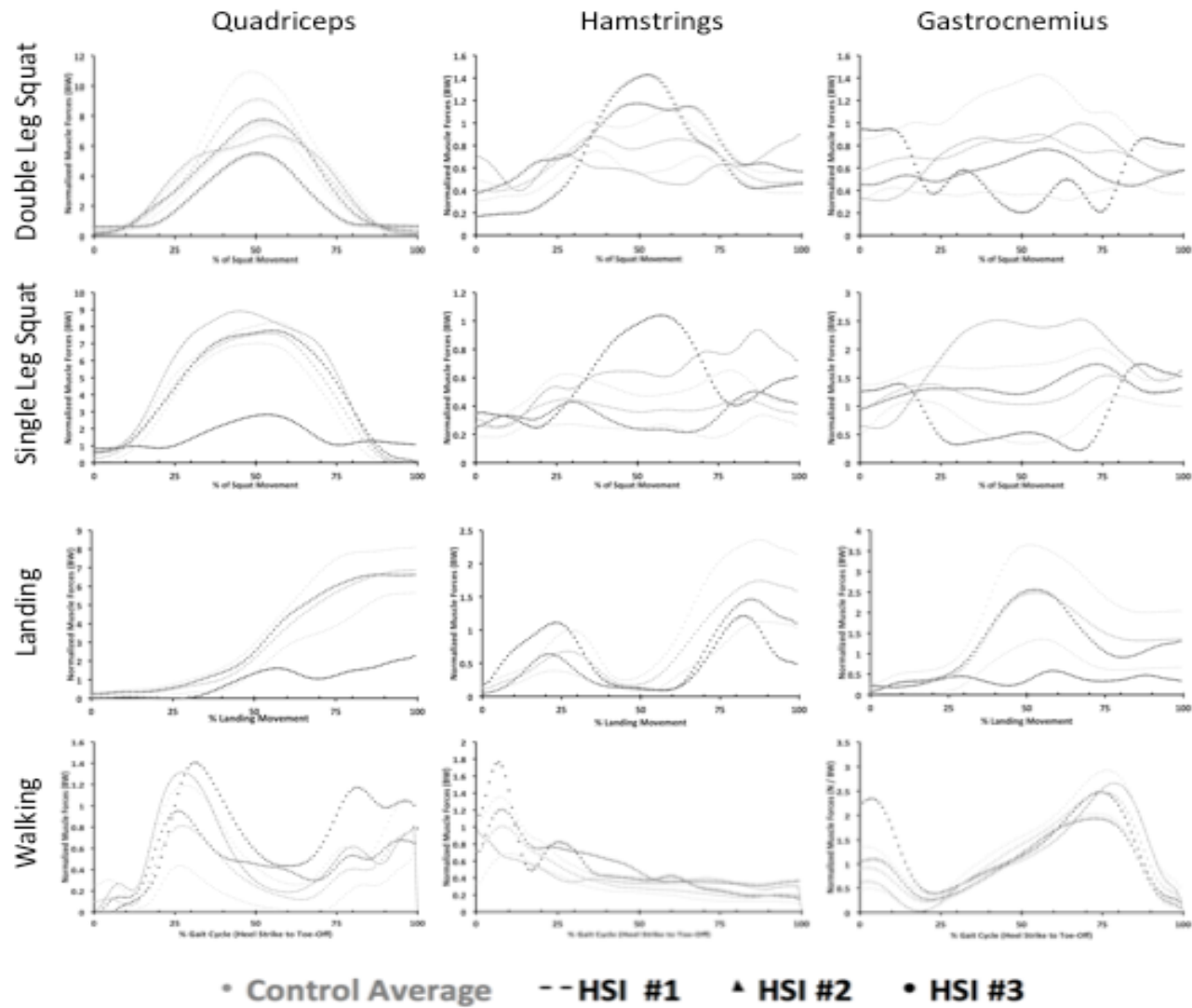


Figure 19: EMG correlation analysis between experimental (black lines) and optimized EMG (grey lines) for all four tasks. The vertical axis is a percentage (%MVC), the right side represents experimental percentages and the left optimized percentages. These are one subjects EMG, therefore it should be looked at as a general representation of how EMG is manipulated by the optimization process

Appendix B: Figure 20 Muscle Forces: Control average vs. previous HSI



Appendix C: IRB Approval



EAST CAROLINA UNIVERSITY
University & Medical Center Institutional Review Board Office
4N-70 Brody Medical Sciences Building Mail Stop 682
600 Moye Boulevard · Greenville, NC 27834
Office 252-744-2914 · Fax 252-744-2284 · www.ecu.edu/irb

Notification of Initial Approval: Expedited

From: Biomedical IRB
To: [Anthony Kulas](#)
CC:
Date: 8/6/2015
Re: [UMCIRB 15-000127](#)
Effect of previous hamstring strains on anterior cruciate ligament loading

I am pleased to inform you that your Expedited Application was approved. Approval of the study and any consent form(s) is for the period of 8/5/2015 to 8/4/2016. The research study is eligible for review under expedited category #4,6,7. The Chairperson (or designee) deemed this study no more than minimal risk.

Changes to this approved research may not be initiated without UMCIRB review except when necessary to eliminate an apparent immediate hazard to the participant. All unanticipated problems involving risks to participants and others must be promptly reported to the UMCIRB. The investigator must submit a continuing review/closure application to the UMCIRB prior to the date of study expiration. The Investigator must adhere to all reporting requirements for this study.

Approved consent documents with the IRB approval date stamped on the document should be used to consent participants (consent documents with the IRB approval date stamp are found under the Documents tab in the study workspace).

The approval includes the following items:

| Name | Description |
|---------------------------|-------------------------------------|
| Hamstring Function Survey | Surveys and Questionnaires |
| INformed consent | Consent Forms |
| Recruitment Announcement | Recruitment Documents/Scripts |
| Study Protocol | Study Protocol or Grant Application |

The Chairperson (or designee) does not have a potential for conflict of interest on this study.

Appendix D: Figure 21 - Subject Specific Ultrasound and Model Based Muscle Parameters

| | | Fmax (N) | OFL (cm) | Penn@OFL (degrees) | TSL (cm) |
|---------------------------------|---------------|------------------|-----------------|---------------------------|-----------------|
| Biceps Femoris Long Head | Control Group | 569.67 ± 239.67 | 11.0 ± 2.0 | 14.57 ± 3.53 | 37.0 ± 3.0 |
| | HSI #1 | 1370.35 | 9.12 | 18.78 | 43.67 |
| | HSI #2 | 382.26 | 11.53 | 13.83 | 34.02 |
| | HSI #3 | 1416.13 | 7.14 | 18.58 | 52.65 |
| Vastus Lateralis | Control Group | 1928.48 ± 419.77 | 11.0 ± 2.0 | 13.62 ± 2.05 | 15.0 ± 1.0 |
| | HSI #1 | 2841.08 | 11.67 | 15.91 | 16.42 |
| | HSI #2 | 1730.08 | 12.80 | 14.08 | 12.21 |
| | HSI #3 | 2832.36 | 10.39 | 13.99 | 20.29 |
| Lateral Gastrocnemius | Control Group | 753.56 ± 233.07 | 5.0 ± 1.0 | 15.90 ± 3.64 | 42.0 ± 3.0 |
| | HSI #1 | 1388.71 | 3.87 | 20.69 | 50.23 |
| | HSI #2 | 681.02 | 4.18 | 16.33 | 37.89 |
| | HSI #3 | 1184.83 | 3.73 | 18.99 | 47.28 |

Figure 21: Model and ultrasound based muscle parameters for control group (mean ± SD) and the three previous hamstring injury subjects. Fmax is the maximum amount of force that the muscle can produce (N). OFL is optimal fiber length (cm). Penn@OFL is the pennation angle at optimal fiber length (degrees). TSL is tendon slack length (cm)

